THE VARIATIONS OF NEUROMUSCULAR EXCITABILITY IN THE COURSE OF ACUTE GLUCIDIC NUTRITIONAL IMBALANCE

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THE VARIATIONS OF NEUROMUSCULAR EXCITABILITY IN THE COURSE OF ACUTE GLUCIDIC NUTRITIONAL IMBALANCE

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ABSTRACT. Neuromuscular excitability during acute glucidic imablance was measured on pigeons. A state of encephalic excitation and an augmentation of the muscular chronaxies were found to result.

The substitution of galactose for glucose in a diet containing 66% of /211
glucides induced the appearance and the rapid evolution of neural problems in the pigeon which are very likely characteristic of those of vitamin B deficiency (convulsive attacks with clonic shaking, contractions, paralytic phenomena) despite the addition of a large dose of vitamin B in the form of dried barm. These manifestations must be attributed to nutritional imbalance, since galactose necessitates for the same reason a very strict balance to be used by the pigeon [1] and more so than lactose. One observes acute glucidic imbalance in the course of this just as in the case of vitamin B deficiency, an augmentation in basal metabolism [2], a lactic impregnation of muscles [3], an appreciable drop of the alcaline reserve [4], and peripheral neural lesions which distinguish themselves from those of vitamin B deficiency because the cylindraxile alteration overcomes the myelinic attack [5].

Since the variations of neuromuscular excitability in the course of total vitamin B deficiency in the pigeon [6], or the B_1 deficiency alone in the rat [7] had already been researched, it seemed interesting to us to

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*Numbers in the margin indicate pagination in the original foreign text.

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study these in the acute glucidic nutritional imbalance of the pigeon. Our measurements were made on adult subjects receiving each day by gavage 20 g of the diet containing 66% of galactose and 4 g of dried barm. According to the customary technique, the animal without having been administered a hypnotic was placed in the normal position in a case allowing the head and feet to be free, with electrodes placed on the undamaged skin. The peripheral neural excitability was measured with a diffused anode consisting of a clip /212
placed on the opposite foot from the one observed, and a cathode formed by a silver loop placed on the extension or flexion motor point of the toes (chronaxies of peripheral nerves), on the course of the sciatic (sensitive chronacy of the cross-flexion reflex), or on the cranial skin (chronaxy of the central motor neurons). Muscular excitability was measured by applying two blunt silver points 1 cm apart on the undamaged skin on a level with the body of the muscle (extensor and flexor of the toes) [8].

The figures of the neural chronaxies stayed at their normal values for 3 to 4 days (generally, chronaxic capacities of 20 mμF for the extension and 40 for the flexion); then suddenly, before all apparent clinical signs, one observes an equalization of chronaxies of the low-level antagonists which lasted until the death of the animal; death took place anywhere between 6 and 9 days. During the period of crisis and great excitation, the figures are very low: 10 or 15; during the calm period, the figures are higher: 20 or 25, but always equalized and the chronaxy of the flexion is well below its normal value. The central chronaxy and the sensitive chronaxy are also a little lower. This variation of peripheral chronaxies is, as we were able to verify completely comparable to that of vitamin B_1 deficiency in the pigeon. This variation is only the repercussion of a central excitation state, more specifically encephalic. By using a nerve section or anesthetizing the animal, one finds, in effect, the chronaxy of a normal constitution which is from 50 to 60 for extension as well as for flexion. Variation, therefore, only permits the state of cerebellar encephalic excitation to be measured (the factor of neural cirses).

The muscular chronaxies stay normal long enough: 60 for the two muscles studied; then, the following day or the day after that up to 300 or 400, one to two days before death. In this instance, therefore, it is undoubtedly not a matter of a direct attack of the muscle, but rather of a secondary degeneration of the muscle fibers whose nerve fibers were altered by the polyneuritic process. If one varies the location of the electrodes on the body of the muscle, one finds, in effect, all the intermediaries of the figures between the normal chronaxies of healthy fibers, which still respond to neural excitation, and the higher chronaxies of the fibers whose nerve has been interrupted. Similar variations are, on the other hand, observable in vitamin B_1 deficiency and diverse experimental types of polyneuritis.

Conclusions — The measurements of neuromuscular excitability practiced on the pigeon in the course of acute glucidic imbalance, which were obtained with the aid of a diet containing 66% galactose in the presence of a large excess of vitamin B, shows the following: First, a state of encephalic excitation comparable to that of vitamin B_1 deficiency and which probably /213
compared with the convulsive neural crises manifested by the animals; second, an augmentation of the muscular chronaxies, indicative of polyneuritic lesions.

Are these two processes, central and peripheral, independent of each other or not? Since the signs of encephalic excitation (variation of neural chronaxies) precede the signs of neural degeneration (variation of muscular chronaxies), one can assume that the central functional problems represent the beginning of polyneuritic lesions [9]. They themselves seem to be dependent on an endogenous intoxication promoted by an acidotic milieu [10].

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